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Is a Type II Endoleak after EVAR a Harbinger of Risk? Causes and Outcome of Open Conversion and Aneurysm Rupture during Follow-up

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Objective. *There is still debate whether type II endoleaks represent a risk for the patient after EVAR. Treatment policies vary from fairly conservative to active intervention. In this analysis risk factors for type II endoleak and adverse events during follow-up were assessed. In addition, risk factors and causes for conversion to open repair and for rupture post-EVAR were studied.*

Methods. *The data of 3595 patients, who underwent operation between 1996 and 2002 in 114 European institutions that collaborated in the EUROSTAR Registry, were assessed. To accurately assess the influence of type II endoleaks patients with type I, III and combined endoleaks were excluded from the present study cohort.*

Results. *A combined adverse outcome event consisting of aneurysmal growth, transfemoral reintervention, and transabdominal secondary procedures (including laparoscopic branch vessel clipping) occurred in 55% in patients with type II endoleak at 3 years, compared to 15% in patients without any endoleak ($p < 0.0001$). Conversion to open repair or post-EVAR rupture was not significantly associated with type II endoleaks. An independent association of device migration and expansion of the aneurysm with late conversion was observed. The cumulative incidence of aneurysm rupture at 3 years of follow-up was 1.2% for an annual rate of 0.4%. Variables that significantly and independently correlated with rupture were size of the aneurysm at preoperative measurement and device migration during follow-up.*

Conclusion. *Endoleak type II may not be harmless as it was more frequently associated with enlargement of the aneurysm and reinterventions. Large aneurysms and migration of the device were the main risk factors for rupture. The clinical implications of these findings may involve more frequent surveillance visits for patients with type II endoleak. Aneurysm expansion is a clear indication for reintervention. Patients with large aneurysms, 65 mm or larger, may also benefit from a more comprehensive surveillance schedule.*

Key Words: Abdominal aortic aneurysm; Endovascular repair; Endoleak; Registry.

Introduction

The feasibility of endovascular abdominal aortic aneurysm repair (EVAR) and the short-term advantages compared to conventional open surgery are no longer in doubt. In patients with suitable aortoiliac anatomy the primary success rate, represented by successful access at endograft deployment, is almost 98%.[1] However, new complications have plagued the stentgraft approach. Failure to totally exclude the AAA from continued perfusion and pressurisation, defined as 'endoleak', remains a potential shortcoming of endoluminal repair,[2,3] and rupture of the aneurysm represents the most dramatic evidence of treatment failure.[4–8] Conversion to open surgery is either performed for manifest rupture of the aneurysm or for impending rupture indicated by radiological or ultrasound studies. In almost any current overview of EVAR the need for intensive surveillance to identify indicators for an increased risk of rupture is emphasised.

Previous assessments, reported from the EUROSTAR collaborators group have demonstrated that the risk of rupture after EVAR, and the need for secondary interventions including conversions to open repair is significantly less in patients with collateral reperfusion (type II) endoleaks than with type I (attachment site endoleak), type III (midgraft leak resulting from disconnection of modular systems or from graft defects), and combined types I and III.[9] Moreover,

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the rate of enlargement of aneurysms appeared distinctly lower in patients with type II endoleaks than with other endoleaks.[10] The general recommendation from the EUROSTAR reports as well as from others is that type I and III endoleaks require prompt repair. The policy in case of type II endoleaks is more open to debate with proponents of early interventions as well as of a conservative approach.[9,11,12] In this latter view the patient should be followed by regular imaging and intervention performed only in case of a significant increase of aneurysm size. With the preferentially non-interventing policy the question remains whether persisting collateral reperfusion endoleak poses the patient to a greater risk of post-EVAR complications such as rupture or secondary interventions than patients without endoleaks. In the present analysis risk factors for isolated type II endoleak and adverse events during follow-up, in particular enlargement of the aneurysm, need for secondary interventions and occurrence of rupture were investigated. In addition risk factors and causes for conversion to open repair and for rupture post-EVAR were assessed. For this analysis an updated and selected portion of the EUROSTAR series was used. As the presence of type I, III and combined endoleaks tend to overtone the effect of other adverse factors in the statistics[8–10,13,14] we excluded patients with these endoleaks from the present study cohort.

Methods

Organisation and patient selection

Details on the organisation of the registry and the data collection have been published in previous articles.[1,8] Clinical events and findings at image studies, most frequently CT examination, were recorded during follow-up at 1, 6, 12, 18, and 24 months and annually thereafter. In the present study a selected portion of the entire EUROSTAR cohort was studied to avoid reiteration of previous analysis. Important exclusion criteria were: (1) all patients of whom the data had been collected retrospectively (enrolment before September 1996), (2) patients treated with the first generation of stentgrafts, as the performance of these older models may not reflect results of the more recent models, (3) patients in whom a type I, III or combined endoleak was observed during follow-up. This category is known to be considerable risk to develop adverse events and the need of prompt reintervention has been extensively emphasised in the existing literature, (4) patients with infrequently used device labels, i.e. less than 100 cases in the database, as small subgroups may preclude a useful analysis. One hundred and fourteen centers contributed the patient data for this study cohort between July 1996 until June 2002 (see Appendix).

Clinical data

All patients had a full medical history and physical examination, contrast enhanced computerised tomography (CT) and angiography. Patients were evaluated with respect to age, sex, smoking, obesity, and fitness for conventional open repair, and physical status classification as defined by the American Society of Anaesthesiologists (ASA). The experience of the surgeon at the type of device used was also evaluated. Other data included in the analysis regarded aneurysm morphology: neck diameter and length, aneurysm diameter, and angulation. The type of aortic device was also used for comparison of outcome events. Endoleak was detected using regular imaging of the abdominal aorta during follow-up. Endograft surveillance was performed using the following imaging techniques: contrast enhanced computer tomography (in 84%), angiography (in 4%), magnetic resonance angiography (in 3%), duplex ultrasound examination (in 8%).

Type II endoleaks were differentiated into persistent and temporary endoleaks. A persistent endoleak was defined as positive identification of endoleak at two occasions with not more than one visit with a negative imaging study in between.

Outcome events: transabdominal interventions included conversions to open surgery, endoscopic clipping of IMA, lumbar and hypogastric arteries, open procedures, related to the infrarenal aorta or iliac arteries.

Aneurysm expansion was defined if the maximum transverse diameter increased by 8 mm or more during any follow visit compared to the preoperative measurement.

Analysis

The clinical features of the patients with endoleak type II were compared to patients without endoleak. Discrete data were analysed using Chi-square test and Fisher correction in case of small subgroups. A multivariate analysis was performed by selecting variables found to be significantly associated with events in the univariate analysis. Continuous variables were compared using the Mann–Whitney U-test. Life table analysis was used to assess incidence rates of adverse events during follow-up. The incidence of

time-dependent variables in patient groups was compared using a log-rank test. In case of multivariate analysis of time dependent variables a Cox proportional hazard model was used. All statistical analyses were performed using Statistical Analysis Software (SAS Institute Inc. 8.0) program (Cary, NC, USA).

Results

Type II endoleaks

Of the overall cohort of 4613 patients, that were included in the EUROSTAR database as of June 2002, 1018 were excluded because of retrospective enrolment, stentgraft models other than AneuRx, Excluder, Talent, Vanguard or Zenith, the presence of a type I, III or any combination of endoleaks during follow-up. This study cohort consisted of 3595 patients, of whom 320 (9%) had an isolated type II endoleak at the 1st month imaging examination, or at any time thereafter (group A), and 3275 (91%) patients who did not have an endoleak at any time during follow-up (group B). The mean duration of follow-up of this cohort was 15 months (0–72).

Preoperative patient characteristics, aorto-iliac morphologic features, operative details, and device brands are summarised in Tables 1 and 2 and significant correlations with type II endoleaks indicated.

It is of note that an ankle-arm blood pressure index of 0.87 or less, current smoking, and poor renal function only in the univariate analysis were associated with a lower prevalence of type II endoleaks (Table 1). Variables that independently correlated with a higher prevalence of type II endoleaks in the multivariate analysis included age of the patient, patent IMA and length of the infrarenal neck (Table 3).

Events during follow-up that occurred significantly more often in patients with type II endoleak were: aneurysm growth ($p = 0.003$, Table 3, Fig. 1), the need for a transfemoral reintervention ($p = 0.001$, Table 3), and transabdominal secondary procedures (only significant in the univariate analysis, $p = 0.02$). Fig. 2 represents these three adverse events as a combined adverse outcome, which occurred far more frequently in patients with type II endoleak ($p < 0.0001$). Late rupture occurred in 12 patients without, and in only one patient with a type II endoleak (not significant). The incidence of late deaths and conversion to open repair was not significantly different in the two study groups.

Conversion to open repair

Conversion to open repair was performed in 71 patients (2%), of whom 45 had an early conversion (within the first month), and 26 a late conversion. The mean period between EVAR and late conversion was 32 months (3–60). The maximum transverse diameter was 61 mm in patients with late conversion and 57 mm in patients without conversion. However, this difference was not significant ($p = 0.09$). Severe angulation of the aneurysm was more frequent in patients with early conversion (23%) than in patients without conversion (11%, $p = 0.018$). Early conversions were more frequently required in the period from 1996 to 1998 than in the period from 1999 to 2002 (2.5% vs. 0.7%, respectively, $p < 0.0001$). The incidence of late conversions was not statistically different for the first and second period regarding the year of the initial EVAR-procedure, although the difference was near the level of significance (4.1% vs. 0.5% at 4 years respectively, $p = 0.056$).

Of the different device brands the use of Talentgrafts correlated with a higher incidence of early conversion ($p = 0.02$). None of the devices were associated with an increased rate of late conversion.

Type II endoleaks were observed during follow-up in 19% of the patients with late conversion compared to 9% of the patients without conversion. This

Table 1. Characteristics, comorbidity, and aorto-iliac morphologic factors.

	Group A, type II endoleak (320 pts)	Group B, no endoleak (3275 pts)	
Age (mean-years, range)	73 (50–93)	71 (37–100)	$p = 0.002$
Male sex	92%	94%	
ASA physical status ≥3	57%	54%	
Ankle/arm systolic blood pressure index ≤0.87	11%	22%	$p = 0.0002$
Current smoking	16%	26%	$p = 0.0002$
Renal insufficiency	15%	20%	$p = 0.02$
Infrarenal neck diameter (mean—mm)	23	23	
Length of infrarenal neck (mean—mm)	29	27	$p = 0.0001$
AAA diameter (mean—mm)	57	57	
Inferior mesenteric artery patent	47%	36%	$p = 0.0004$

Variables without indicated *p*-value had a $p > 0.05$.

Table 2. Device brands, operative details, and findings at completion angiography.

	Group A, type II endoleak (320 pts)	Group B, no endoleak (3275 pts)	
AneuRx	77 (9%)	756 (91%)	
Excluder	35 (10%)	314 (90%)	
Talent	45 (6%)	730 (94%)	$p = 0.0006$
Vanguard	94 (12%)	665 (88%)	$p = 0.0001$
Zenith	69 (8%)	810 (92%)	
Embolisation of side branches	13%	9%	$p = 0.016$
Blocking of one or two hypogastric arteries	22%	16%	$p = 0.016$
Reperfusion endoleak, lumbar or IMA	21%	7%	$p = 0.0001$

Variables without indicated p-value had a $p > 0.05$.

difference was not significant. In addition, the observation of proximal, midgraft and distal endoleaks during follow-up did not correlate with conversion.

Observed events during follow-up, that correlated in the univariate analysis with the need for late conversion included stenosis or thrombosis of the device (in 23% of patients with late conversion, $p = 0.02$), migration of the device (in 54%, $p < 0.0001$), aneurysm expansion (in 25%, $p = 0.017$), and rupture of the aneurysm (in 23%, $p < 0.0001$). For migration, aneurysm expansion an independent association with late conversion was observed (Table 3).

The first-month mortality associated with early conversion was 13% (6/45) compared to 2.3% in patients without conversion ($p < 0.0001$). Mortality during the entire follow-up period in patients with late conversion was 7.7% (2/26) compared to 7.4% (261/3524) in patients without conversion (ns).

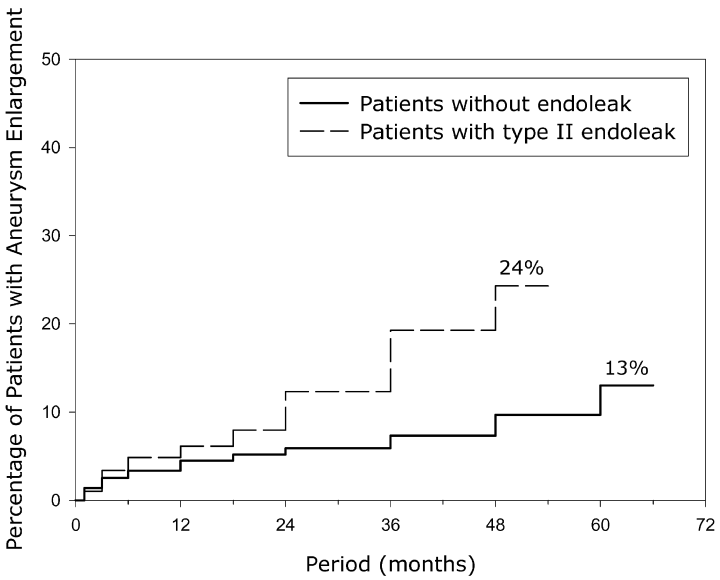
Rupture of the aneurysm

In this study cohort 14 patients experienced rupture of the aneurysm after EVAR. Rupture occurred at the mean of 24 months (0–48) from the initial operation. The cumulative incidence of rupture at 3 years of follow-up was 1.2%. Eight of the 14 patients had preoperatively a diameter of 65 mm or greater and this diameter had not decreased at the time of rupture in five of these patients. Of the variables assessed at baseline by univariate analysis the maximum aneurysm diameter correlated significantly with post-EVAR rupture (70 mm (55–105) in patients with rupture vs. 57 mm, (30–145), $p = 0.004$). Significant angulation of the infrarenal neck and associated common iliac aneurysm had borderline significant correlations with rupture ($p = 0.05$ and 0.03, respectively). At multivariate analysis the preoperative aneurysm diameter was the only independent correlating variable (Table 3). The year in which EVAR was performed had no correlation with the risk of rupture. None of the endograft models was associated with an increased risk of rupture.

Of follow-up data migration of the device correlated significantly with rupture (in 29% of patients with rupture vs. 3.2%, $p = 0.0017$). There was a weak correlation between thrombosis and stenosis of devices with rupture ($p = 0.03$). No correlation between endoleak type II and aneurysm was demonstrated. In the multivariate model migration was the

Table 3. Multivariate analysis of type II endoleak, late conversion and post-EVAR aneurysm rupture.

Event	Category of variables	Variable with independent correlation	p-value	Confidence interval
Type II endoleak	Baseline	Age of patient	0.001	1.01–1.06
		Preoperative patency of IMA	0.031	1.03–1.99
		Length of infrarenal neck	0.006	1.01–1.03
		Current smoking	0.008	0.38–0.87
		Ankle–arm BP index ≤0.87	0.0007	0.23–0.68
	Operation	Coil embolisation of side branches	0.014	1.10–2.30
		Patent IMA or lumbar arteries on operative arteriogram	<0.0001	2.21–3.82
	Follow-up	Expansion of aneurysm	0.003	1.20–2.51
		Transfemoral reintervention	<0.0001	2.52–4.58
Late conversion to open repair	Follow-up	Device migration	<0.0001	4.11–21.35
		Expansion of aneurysm	0.024	1.16–8.32
		Aneurysm rupture	<0.0001	6.79–50.20
Rupture of aneurysm	Baseline	Aneurysm diameter	<0.0001	1.04–1.09
	Follow-up	Device migration	0.019	1.26–13.37



Patients at risk	0	12	24	36	48	60	months
without endoleak	2340	1642	940	522	191	26	
with endoleak type II	296	214	121	58	15	-	

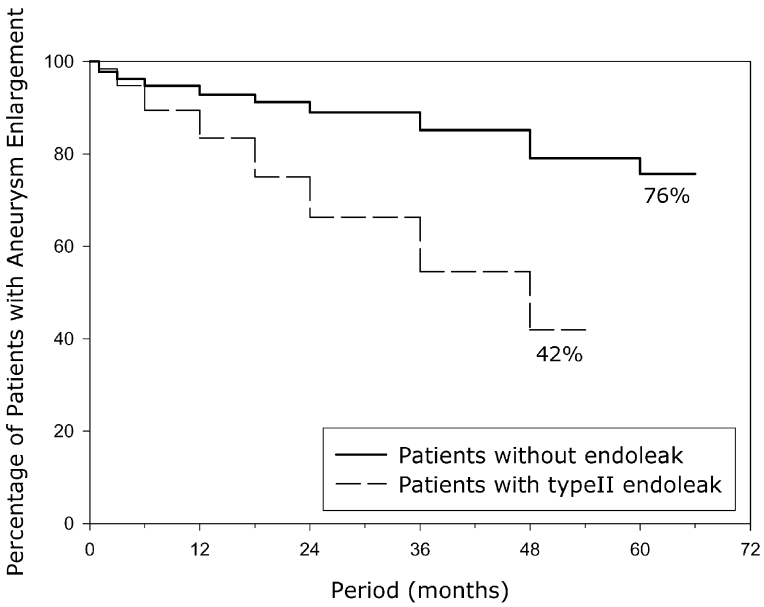
Fig. 1. Incidence of aneurysm enlargement in patients with and without type II endoleak. The difference is significant ($p = 0.003$).

only variable, that maintained a significant correlation with rupture (Table 3).

Eight of the patients with rupture died in association with post-EVAR rupture, for a mortality of 57%, compared to a combined early and late mortality rate of 10% in patients without rupture ($p < 0.0002$).

Discussion

The less invasive nature of the procedure and the generally satisfactory early results, have made endovascular AAA-repair an appealing therapeutic alternative to most patients with AAA. Studies that compared the outcome of EVAR with open surgery have consistently demonstrated a significant reduction in morbidity with endovascular repair.[15–18] In a recent report by Arko *et al.* aneurysm related death showed a difference of 5% combined early and late aneurysm related death at 3 years of follow-up in favour of EVAR.[19] This difference nearly reached statistical significance. However, this study, like previous comparative studies, was not randomised and patient selection may have skewed the outcome in the study groups in one or another way. Although the prospects of EVAR look exceedingly bright at this time, recognition of risk factors for adverse events, such as the need for conversion to open surgery and late post-EVAR rupture, may lead to further improvement of the techniques. In previous publications, based on the EUROSTAR registry, risk factors for proximal device migration and device-related endoleaks type I and III have been reported.[9,10,20,21] In the present study, the significance of the most common endoleak, type II, was assessed in greater detail, with regard to severe outcome events. To make the conclusions of the study more applicable to current practice, retrospectively enrolled patients, in whom predominantly endografts were used that are not available for many years were also excluded from the analysis. In these previous publications the effect of endoleaks other than type II and factors typically associated with older graft versions on conversion or rupture were quite dominant.[8,10,14] The present modifications in the composition of the study cohort were intentionally made to highlight the effect of other risk factors. It is felt in the EUROSTAR organisation that a continuous updating and renewal of analysis are most valuable to increase insight in the technique, and assumingly may improve guidelines for patient selection and surveillance.



Patients at risk	0	12	24	36	48	60 months
without endoleak	2548	1671	908	491	170	22
with endoleak type II	304	194	91	37	10	-

Fig. 2. Incidence of combined outcome event consisting of aneurysm enlargement, transfemoral and transabdominal secondary intervention (with univariate comparison) in patients with and without type II endoleak. The difference is significant ($p =< 0.0001$).

Of the preoperative patient and morphologic factors age of the patient, length of the infrarenal neck and patency of IMA were shown to be associated with increased occurrence of type II endoleak. Except for the latter condition one may only speculate on the biologic basis for these correlations. The reversed association (type II endoleaks were less frequently observed) of ABI ≤ 0.87, and current smoking is surprising and had not been observed previously. One may reason that these factors are markers of peripheral arterial occlusive disease, perhaps associated with more severe atherosclerosis of side branch vessels. This condition may lead to more frequent occlusion of collaterals compared to patients with more distensible lumbar, hypogastric and inferior mesenteric arteries. Moreover, current smoking is known to cause changes in the coagulation profile of the blood,[22] which also may influence the tendency of spontaneous occlusion of small vessels. This reasoning seems in agreement with the adverse effect of therapeutic Warfarin in spontaneous closure of type II endoleak as was recently documented by Fairman *et al.*[23]

Some groups have adopted an aggressive approach to type II endoleaks in that persisting leaks are coil embolised, irrespective whether any enlargement of the aneurysm sac is observed.[11,24] The rational behind this approach is based on *in vitro* experiments, in which open collateral vessels maintained systemic pressure within an excluded sac.[25] Several clinical studies also have suggested that aneurysm growth and even rupture of excluded aneurysms, surgically or by stentgraft, can be caused by patent collateral pathways.[7,26,27]

In the present study we demonstrated that a patent IMA at preoperative imaging was significantly associated with late collateral endoleak. Other groups made similar observations of preoperative branch vessel patency and increased rates of type II endoleak following EVAR.[28,29] Considering these observations, prophylactic branch embolisation before or during EVAR may seem a rational approach to avoid type II endoleak development. However, no reduction of endoleaks was documented in a number of studies in which this approach was attempted.[30,31] In the present study coil embolisation before or during EVAR correlated with an increased rate of type II endoleaks. This finding supports the supposition that complete

Table A1. Participants from 114 institutions who contributed the data of this EUROSTAR Study.

Austria		Norway	
Vienna	Prof. G. Kretschmer	Oslo	Prof. A. Kroeze
Belgium		Oslo	Dr K. Krohg-Soerensen
Aalst	Dr J. de Coster	Trondheim	Prof. H. Myhre
Aalst	Dr Degrieck	Poland	
Antwerpen/Wilrijk	Dr M. van Betsbrugge	Lublin	Prof. J. Michalak
Assebroek/Brugge	Dr H. Tubbax	Warsaw	Dr M.L. Nowicki
Bonheiden	Dr P.M.A.J. Peeters	Spain	
Bruxelles	Dr R. Verhelst	Barcelona	Dr V. Riambau
Gilly	Dr H. Massin	Barcelona	Dr M. Cairols
Kortrijk	Dr L. van Lysebeth	Barcelona	Dr J. Escudero Rodriguez
Leuven	Prof. A. Nevelsteen	Donostia San Sebastian	Dr M. de Blas
Lommel	Dr M. Mattens	La Coruna	Dr R. Segura
Mechelen	Dr Y. Tielemans	Leon	Dr R. Fernandez-Samos Gutierrez
St.Truiden	Dr F. van Elst	Lugo	Dr J.R. Pulpeiro
St.Truiden	Dr L. Verougstraete	Madrid	Dr D. Tagarro
Turnhout	Dr P. Stabel	Madrid	Dr E. Criado
Vilvoorde	Dr E. Sebrechts	Madrid	Dr Sanchez-Corral
Denmark		Madrid	Dr J. Urbano
Copenhagen	Dr L. Jörgensen	Madrid	Dr F. Acin
Odense	Dr P. Justesen	Pamplona	Dr L. Fernandez Alonso
France		Valladolid	Dr V. Gutiérrez Alonso
Draguignan	Dr C. Mialhe	Sweden	
Grenoble	Dr J.L. Magne	Lund	Prof. L. Norgren
Lille Cedex	Dr M.A. Vasseur	Orebro	Dr Th. Nordh Larzon
Lyon	Dr B. Age	Switzerland	
Lyon	Dr P. Feugier	Bern	Dr J. Schmidli
Marseille	Dr Ph. Piquet	Zurich	Dr M. Enzler
Montpellier	Prof. C. Marty-Ane	The Netherlands	
Nancy	Dr C. Amicabile	Alkmaar	Dr H.A. van Dijk
Nanterre	Dr J. Marzella	Amsterdam	Dr R. Balm
Nimes	Dr Y. Cardon	Amsterdam	Dr W. Wisselink
Paris cedex	Prof. J.C. Gaux	Amsterdam	Dr Vahl
Paris Creteil Cedex	Prof. J.P. Becquemin	Apeldoorn	Dr E.G.J. Vermeulen
St Etienne	Prof. J.P. Favre	Arnhem	Dr W.R. de Vries
St Laurant du Var	Prof. P. Kreitmann	Delft	Dr J. Koning
Toulouse	Prof. H. Rousseau	Den Haag	Dr J.C.A. de Mol van Otterloo
Toulouse cedex	Dr C. Giraud	Den Haag	Dr H. van Overhagen
Germany		Dordrecht	Dr R.P. Tutein Nolthenius
Bonn	Dr A. Viehofer	Eindhoven	Dr J. Buth
Dusseldorf	Dr R. Kolvenbach	Enschede	Dr R.H. Geelkerken
Frankfurt	Prof. W. Stelter	Geldrop	Dr F.Th.P.M. van der Linden
Frankfurt	Prof. H. Sievert	Groningen	Dr E. Verhoeven
Freiburg	Dr P. Uhrmeister	Groningen	Dr H.R. Dop
Hamburg	Prof. H. Kortmann	Maastricht	Dr G.W.H. Schurink
Hanover	Dr G. Voshage	Nieuwegein	Dr F. Moll
Kempten	Dr Antoni	Nijmegen	Dr W.B. Barendrecht
Koblenz	Dr R. Wickenhöfer	Nijmegen	Prof. J. van Vliet
Mainz	Dr C. Duber	Rotterdam	Dr A. de Smet
Marburg	Dr H. Alfke	Rotterdam	Dr M. van Sambeek
Munich	Prof. P.C. Maurer	Rotterdam	Dr A.C. van der Ham
Oldenburg	Dr C. Ratusinski	Tilburg	Dr Hamming
Ulm	Dr Pamler	Tilburg	Dr S. Kranendonk
Greece		Utrecht	Dr J. Blankensteijn
Psihico Athens	Prof. P. Balas	Veldhoven	Dr J.A. Charbon
Ireland		Zwolle	Dr P. Jörning
Dublin	Prof. Shanik	United Kingdom	
Israel		Bournemouth	Dr S. Darke
Tel Aviv	Prof. B. Morag	Bristol	Dr R. Baird
Italy		Chester	Dr G. Abbott
Perugia	Prof. P. Cao	Glasgow	Dr R. Edwards
Roma	Dr M. Scoccianti	Hull	Dr D. Ettler
Luxembourg		Liverpool	Dr P. Harris
Luxembourg	Dr P. Berg	London	Dr J. Wolfe
Monaco		Manchester	Dr R. Asleigh
Monaco	Dr C. Mialhe	New Castle-Upon-Tyne	Dr M.G. Wyatt

obliteration of all patent branches often fails. Whether prophylactic (at the time of EVAR) rather than therapeutic coil embolisation of endoleaks will result in a durable reduction of blood pressure within the aneurysmal sac remain indeterminate. Some animal experiments showed that pressures within the aneurysmal sac remained unchanged after coil implantation.[32] However, the same group demonstrated later in an *in vivo* model that long narrow channels mimicking collateral vessels could be obliterated more effectively by catheter intervention than channels representing device attachment related endoleaks.[33] Whatever the outcome of these experiments, several studies including the present one, do not suggest that secondary intervention by catheter technique or laparoscopic clipping (a recently popular alternative) are indicated for type II endoleaks unless the aneurysm clearly shows expansion.[9,10,34]

Enlargement of the aneurysm is often considered evidence of treatment failure. While other reports often use a 5 mm diameter increase on CT, as the threshold to define growth, we considered 8 mm or greater as the threshold to indicate a significant change. It was assumed that the interobserver variability in a registry, without use of core-lab monitoring, would be larger than in single institutional series. In patient without endoleak freedom-from-enlargement of the aneurysm was observed in 93% at 3 years, while the incidence was 81% in patients with type II endoleak. These findings are unique for the EUROSTAR database, primarily because other studies reporting on aneurysm size changes, made no distinction between the different types of endoleaks.[10,35,36]

Early conversion was substantially less frequently performed after 1998 considering a rate of 0.7% compared to 2.5% in the previous years. This reflects a changing attitude towards the indication to resolve intraoperative complications, such as access problems and device migration during EVAR. Reports from May *et al.* and subsequent studies including one by the EUROSTAR group, have emphasised the high mortality associated with primary conversion.[14,37] Late conversions were primarily performed for rupture, migration or aneurysm enlargement.[14] Although conversions were more frequently performed in patients with type II endoleak than in patients without endoleak, this difference was not significant. The mortality associated with late conversions was 8% (2/26), which compared favourably with other current multicenter series.[38]

The proportion of patients experiencing post-EVAR rupture in the study cohortiously influenced by the exclusion of patients with type I and III endoleaks. In addition this rate is dependent of the duration of follow-up. The entire EUROSTAR cohort (4613) including patients with early stentgraft versions, retrospectively enrolled patient data and all types of endoleaks involved 37 patients with rupture (0.8%) as of June 2002. Details on this cohort will be published shortly. The report by Harris *et al.*, which was submitted in March 2000, involved 14 patients out of 2464 with aneurysm rupture, for an annual cumulative rupture rate of approximately 1%.[8] In the present study the annual rate of rupture was 0.4. However, the patient selection described above makes these figures incomparable. The overall mortality after rupture was 57%, a figure that is fairly typical for ruptured AAA. With regard to the cause of rupture it was not surprising that the preoperative aneurysm diameter and migration during follow-up correlated independently with post-EVAR aneurysm rupture. Migration of endografts may lead to sudden pressurisation and rupture of aneurysm sacs.

Conclusion

A selected patient cohort was examined excluding the influence of type I and III endoleaks, which have a notorious reputation to be associated with adverse outcome. The clinical implications of this study are several. Endoleak type II may not be harmless, as it was more frequently associated with enlargement of the aneurysm and reinterventions. Our findings suggest that more frequent surveillance examinations are indicated than in patients without collateral endoleak. The indication for reintervention is primarily dictated by documented aneurysm expansion. Primary conversion is now generally perceived to be a dangerous procedure, and its use has been markedly reduced in the recent years. Large aneurysms and migration of the device were the main risk factors for rupture. More comprehensive surveillance of aneurysms of 65 mm or larger and prompt recognition and treatment of device migration are mandatory.

Appendix

Table A1

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